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(FILE 'HOME' ENTERED AT 13:57:34 ON 01 APR 2002)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCPLUS, NTIS,
LIFESCI' ENTERED AT 13:57:58 ON 01 APR 2002

L1 5706 S CORYNEBACTERIUM (A)GLUTAMICUM
L2 0 S PHOSPHOENYL PYRUVATE (W) SUGAR (W) PHOSPHOTRANSFERASE?
L3 3480 S "SUGAR PHOSPHOTRANSFERASE?"
L4 11 S PHOSPHOENYL PYRUVATE
L5 16 S L1 AND L3
L6 10 DUP REM L5 (6 DUPLICATES REMOVED)
L7 19911 S "PTS"
L8 1406 S L3 AND L7
L9 152465 S PYRUVATE
L10 146 S L8 AND L9
L11 0 S L1 AND L10
L12 11661 S BREVIBACTERIUM
L13 5 S L12 AND L10
L14 2 DUP REM L13 (3 DUPLICATES REMOVED)
L15 473 S L3 AND "PEP"
L16 280 S L15 AND L7
L17 48 S L9 AND L16
L18 24 DUP REM L17 (24 DUPLICATES REMOVED)
L19 0 S L18 AND L1
E POMPEJUS M/AU
L20 26 S E3
L21 48 S E3-E4
E KROGER B/AU
L22 92 S E3
E SCHRODER H/AU
L23 1910 S E3
E ZELDER O/AU
L24 180 S E3
E HABERHAUER G/AU
L25 236 S E3-E6
L26 2444 S L21 OR L22 OR L23 OR L24 OR L25
L27 0 S L26 AND L17
L28 2 S L26 AND L3

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NEWS 3 Jan 25 Searching with the P indicator for Preparations
NEWS 4 Jan 29 FSTA has been reloaded and moves to weekly updates
NEWS 5 Feb 01 DKILIT now produced by FIZ Karlsruhe and has a new update frequency
NEWS 6 Feb 19 Access via Tymnet and SprintNet Eliminated Effective 3/31/02
NEWS 7 Mar 08 Gene Names now available in BIOSIS
NEWS 8 Mar 22 TOXLIT no longer available
NEWS 9 Mar 22 TRCTHERMO no longer available
NEWS 10 Mar 28 US Provisional Priorities searched with P in CA/CAplus and USPATFULL
NEWS 11 Mar 28 LIPINSKI/CALC added for property searching in REGISTRY

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FILE 'LIFESCI' ENTERED AT 13:57:58 ON 01 APR 2002
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=> s corynebacterium (a)glutamicum

L1 5706 CORYNEBACTERIUM (A) GLUTAMICUM

=> s phosphoenolpyruvate (w)sugar (w)phosphotransferase?

L2 0 PHOSPHOENOLPYRUVATE (W) SUGAR (W) PHOSPHOTRANSFERASE?

=> s "sugar [phosphotransferase?]"

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SEARCH ENDED BY USER

=> s "sugar phosphotransferase?"

L3 3480 "SUGAR PHOSPHOTRANSFERASE?"

=> s phosphoenolpyruvate

L4 11 PHOSPHOENOLPYRUVATE

=> s 11 and l3

L5 16 L1 AND L3

=> dup rem 15

PROCESSING COMPLETED FOR L5

L6 10 DUP REM L5 (6 DUPLICATES REMOVED)

=> d 1-10 ibib ab

L6 ANSWER 1 OF 10 BIOTECHDS COPYRIGHT 2002 DERWENT INFO AND ISI
ACCESSION NUMBER: 2001-04894 BIOTECHDS

TITLE: **Corynebacterium glutamicum** nucleic acids
encoding phosphoenolpyruvate:**sugar**
phosphotransferase system proteins or their portions,
useful for typing or identifying *C. glutamicum* or related
bacteria, and as markers for transformation;
selectable marker

AUTHOR: Pompejus M; Kroeger B; Schroeder H; Zelder O; Haberhauer G

PATENT ASSIGNEE: BASF

LOCATION: Ludwigshafen, Germany.

PATENT INFO: WO 2001002583 11 Jan 2001

APPLICATION INFO: WO 2000-DE973 27 Jun 2000

PRIORITY INFO: DE 1999-1042097 3 Sep 1999; US 1999-142691 1 Jul 1999

DOCUMENT TYPE: Patent

LANGUAGE: English

OTHER SOURCE: WPI: 2001-080989 [09]

AB Isolated **Corynebacterium glutamicum** ATCC 13032
nucleic acids encoding phosphoenolpyruvate:**sugar**-
phosphotransferase system (PTS) proteins or their fragments are
claimed. A PTS nucleic acid (N1) does not consist of any of the
F-designated genes defined and is selected from one of 17 disclosed
nucleic acid sequences (S1) and their fragments nucleic acid which encode

a protein selected from one of the 17 protein sequences (S2) disclosed; nucleic acid encoding a naturally occurring allelic variant of a protein selected from (S2). Also claimed are methods for producing the proteins; *C. glutamicum* PTS protein and its fragments; diagnosis of *Corynebacterium diphtheriae* infection; fusion proteins; antisense PTS nucleic acid; a method for screening molecules which modulate the activity of a PTS protein; and a transformed host cell. (144pp)

L6 ANSWER 2 OF 10 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2001:31659 HCAPLUS

DOCUMENT NUMBER: 134:96287

TITLE: ***Corynebacterium glutamicum* genes
encoding phosphoenolpyruvate:sugar
phosphotransferase system proteins**

INVENTOR(S): Pompejus, Markus; Kroger, Burkhard; Schroder, Hartwig;
Zelder, Oskar; Haberhauer, Gregor

PATENT ASSIGNEE(S): BASF Aktiengesellschaft, Germany

SOURCE: PCT Int. Appl., 143 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 6

PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|------------------------|---|----------|--------------------|------------|
| WO 2001002583 | A2 | 20010111 | WO 2000-IB973 | 20000627 |
| WO 2001002583 | A3 | 20010726 | | |
| W: | AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,
HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,
LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU,
ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM | | | |
| RW: | GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
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CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG | | | |
| PRIORITY APPLN. INFO.: | | | US 1999-142691P | P 19990701 |
| | | | US 1999-150310P | P 19990823 |
| | | | DE 1999-19942095 A | 19990903 |
| | | | DE 1999-19942097 A | 19990903 |

AB Isolated nucleic acid mols., designated phosphoenolpyruvate:sugar phosphotransferase (PTS) nucleic acid mols., which encode novel PTS proteins from *Corynebacterium glutamicum* are described. The invention also provides antisense nucleic acid mols., recombinant expression vectors contg. PTS nucleic acid mols., and host cells into which the expression vectors have been introduced. The invention still further provides isolated PTS proteins, mutated PTS proteins, fusion proteins, antigenic peptides and methods for the improvement of prodn. of a desired compd. from *C. glutamicum* based on genetic engineering of PTS genes in this organism.

L6 ANSWER 3 OF 10 MEDLINE

DUPPLICATE 1

ACCESSION NUMBER: 2001700841 MEDLINE

DOCUMENT NUMBER: 21617011 PubMed ID: 11741338

TITLE: The ptsI gene encoding enzyme I of the phosphotransferase system of *Corynebacterium glutamicum*.

AUTHOR: Kotrba P; Inui M; Yukawa H

CORPORATE SOURCE: Research Institute of Innovative Technology for the Earth,
9-2, Kizugawadai, Kizu-cho, Soraku-gun, Kyoto, 619-0292,
Japan.

SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (2001

Dec 21) 289 (5) 1307-13.
Journal code: 0372516. ISSN: 0006-291X.

PUB. COUNTRY: United States
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200202
ENTRY DATE: Entered STN: 20011220
Last Updated on STN: 20020220
Entered Medline: 20020219

AB The phosphoenolpyruvate:carbohydrate phosphotransferase system (PTS) is widespread among bacteria where it mediates carbohydrate uptake and often serves in carbon control. Here we present cloning and analysis of the monocistronic ptsI gene of **Corynebacterium glutamicum** R, which encodes PTS Enzyme I (EI). EI catalyzes the first reaction of PTS and the reported ptsI was shown to complement the corresponding defect in *Escherichia coli*. The deduced 59.2-kDa EI of 564 amino acids shares more than 50% homology with EIs from *Bacillus stearothermophilus*, *Bacillus subtilis*, and *Lactobacillus sake*. Chromosomal inactivation of ptsI demonstrated that EI plays an indispensable role in PTS of *C. glutamicum* R and this system represents a dominant sugar uptake system. Cellobiose was only transported and utilized in adaptive mutants of *C. glutamicum* R. Cellobiose transport was also found to be PTS-dependent and repressed by PTS sugar glucose.

L6 ANSWER 4 OF 10 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2001274434 MEDLINE
DOCUMENT NUMBER: 21258526 PubMed ID: 11361073
TITLE: **Corynebacterium glutamicum**: a dissection of the PTS.
AUTHOR: Parche S; Burkovski A; Sprenger G A; Weil B; Kramer R; Titgemeyer F
CORPORATE SOURCE: Lehrstuhl fur Mikrobiologie, Friedrich-Alexander-Universitat Erlangen-Nurnberg, Germany.
SOURCE: JOURNAL OF MOLECULAR MICROBIOLOGY AND BIOTECHNOLOGY, (2001 Jul) 3 (3) 423-8.
Journal code: DSF; 100892561. ISSN: 1464-1801.
PUB. COUNTRY: England: United Kingdom
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200110
ENTRY DATE: Entered STN: 20011022
Last Updated on STN: 20011022
Entered Medline: 20011018

AB The high-GC Gram-positive actinomycete **Corynebacterium glutamicum** is commercially exploited as a producer of amino acids that are used as animal feed additives and flavor enhancers. Despite its beneficial role, carbon metabolism and its possible influence on amino acid metabolism is poorly understood. We have addressed this issue by analyzing the phosphotransferase system (PTS), which in many bacteria controls the flux of nutrients and therefore regulates carbon metabolism. The general PTS phosphotransferases enzyme I (EI) and HPr were characterized by demonstration of PEP-dependent phosphotransferase activity. An EI mutant exhibited a pleiotropic negative phenotype in carbon utilization. The role of the PTS as a major sugar uptake system was further demonstrated by the finding that glucose and fructose negative mutants were deficient in the respective enzyme II PTS permease activities. These carbon sources also caused repression of glutamate uptake, which suggests an involvement of the PTS in carbon regulation. The observation that no HPr kinase/phosphatase could be detected suggests that the mechanism of carbon regulation in *C. glutamicum* is different to the

one found in low-GC Gram-positive bacteria.

L6 ANSWER 5 OF 10 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.DUPLICATE 3
ACCESSION NUMBER: 2000015847 EMBASE
TITLE: Cloning, nucleotide sequencing, and characterization of the ptsG gene encoding glucose-specific enzyme II of the phosphotransferase system from *Brevibacterium lactofermentum*.
AUTHOR: Yoon K.-H.; Lee K.-N.; Lee J.-K.; Park S.C.
CORPORATE SOURCE: K.-H. Yoon, School of Food Biotechnology, Woosong University, San 7-6, Jayang-Dong, Dong-Gu, Taejon 300-100, Korea, Republic of. ykh@lion.woosong.ac.kr
SOURCE: Journal of Microbiology and Biotechnology, (1999) 9/5 (582-588).
Refs: 31
ISSN: 1017-7825 CODEN: JOMBES
COUNTRY: Korea, Republic of
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
LANGUAGE: English
SUMMARY LANGUAGE: English
AB A *Brevibacterium lactofermentum* gene coding for a glucose-specific permease of the phosphoenolpyruvate-dependent phosphotransferase system (PTS) was cloned, by complementing an *Escherichia coli* mutation affecting a ptsG gene with the *B. lactofermentum* genomic library, and completely sequenced. The gene was identified as a ptsG, which enables an *E. coli* transformant to transport non-metabolizable glucose analogue 2-deoxyglucose (2DG). The ptsG gene of *B. lactofermentum* consists of an open reading frame, of 2,025 nucleotides encoding a polypeptide of 674 amino acid residues and a TAA stop codon. The 3' flanking region contains two stem-loop structures which may be involved in transcriptional termination. The deduced amino acid sequence of the *B. lactofermentum* enzyme II(Glc) specific to glucose (EII(Glc)) has a high homology with the *Corynebacterium glutamicum* enzyme II(Man) specific to glucose and mannose (EII(Man)), and the *Brevibacterium ammoniagenes* enzyme II(Glc) specific to glucose (EII(Glc)). The 171-amino-acid C-terminal sequence of the EII(Glc) is also similar to the *Escherichia coli* enzyme IIA(Glc) specific to glucose (IIA(Glc)). It is interesting that the arrangement of the structural domains, IIBCA, of the *B. lactofermentum* EII(Glc) protein is identical to that of EIIs specific to sucrose or beta-glucoside. Several *in vivo* complementation studies indicated that the *B. lactofermentum* EII(Glc) protein could replace both EII(Glc) and EIIA(Glc) in an *E. coli* ptsG mutant or crr mutant, respectively.

L6 ANSWER 6 OF 10 HCPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:714378 HCPLUS
DOCUMENT NUMBER: 130:109230
TITLE: L-Lysine experimental yields by *Corynebacterium glutamicum* on carbon substrates
AUTHOR(S): Ruklisha, M.; Ionina, R.
CORPORATE SOURCE: Institute of Microbiology and Biotechnology, University of Latvia, Riga, LV-1586, Latvia
SOURCE: Meded. - Fac. Landbouwkld. Toegepaste Biol. Wet. (Univ. Gent) (1998), 63(4a), 1341-1344
CODEN: MFLBER; ISSN: 1373-7503
PUBLISHER: Universiteit Gent, Faculteit Landbouwkundige en Toegepaste Biologische Wetenschappen
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Growth of *C. glutamicum* and *C. flavum* on different carbon and energy substrates was investigated to det. the advantages of definitive

substrates for increasing lysine prodn. and to identify rate-limiting metabolic steps. The impact of the C source on the growth rate, sugar uptake, lysine yield, and catabolic and lysine-synthesizing enzyme activities was detd. Sucrose was the best C source for increased lysine prodn; an equimolar mix. of glucose and fructose was effective also. The obsd. metabolic differences resulted in differences in metabolite overflow in the direction of lysine synthesis.

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 7 OF 10 MEDLINE

ACCESSION NUMBER: 1998314508 MEDLINE

DOCUMENT NUMBER: 98314508 PubMed ID: 9652400

TITLE: Carbon-flux distribution in the central metabolic pathways of *Corynebacterium glutamicum* during growth on fructose.

AUTHOR: Dominguez H; Rollin C; Guyonvarch A; Guerquin-Kern J L; Cocaign-Bousquet M; Lindley N D

CORPORATE SOURCE: Centre de Bioingenierie Gilbert Durand, UMR CNRS/INSA & L.A. INRA, Institut National des Sciences Appliquees, Complexe Scientifique de Rangueil, Toulouse, France.

SOURCE: EUROPEAN JOURNAL OF BIOCHEMISTRY, (1998 May 15) 254 (1) 96-102.

PUB. COUNTRY: Journal code: EMZ; 0107600. ISSN: 0014-2956.
GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199807

ENTRY DATE: Entered STN: 19980811
Last Updated on STN: 19990129
Entered Medline: 19980728

AB Growth of *Corynebacterium glutamicum* on fructose was significantly less than that obtained on glucose, despite similar rates of substrate uptake. This was in part due to the production of overflow metabolites (dihydroxyacetone and lactate) but also to the increased production of CO₂ during growth on fructose. These differences in carbon-metabolite accumulation are indicative of a different pattern of carbon-flux distribution through the central metabolic pathways. Growth on glucose has been previously shown to involve a high flux (> 50% of total glucose consumption) via the pentose pathway to generate anabolic reducing equivalents. NMR analysis of carbon-isotope distribution patterns of the glutamate pool after growth on 1-¹³C- or 6-¹³C-enriched fructose indicates that the contribution of the pentose pathway is significantly diminished during exponential growth on fructose with glycolysis being the predominant pathway (80% of total fructose consumption). The increased flux through glycolysis during growth on fructose is associated with an increased NADH/NAD⁺ ratio susceptible to inhibit both glyceraldehyde-3-phosphate dehydrogenase and pyruvate dehydrogenase, and provoking the overflow of metabolites derived from the substrates of these two enzymes. The biomass yield observed experimentally is higher than can be estimated from the apparent quantity of NADPH associated with the pentose pathway and the flux through isocitrate dehydrogenase, suggesting an additional reaction yielding NADPH. This may involve a modified tricarboxylic acid cycle involving malic enzyme, expressed to significantly higher levels during growth on fructose than on glucose, and a pyruvate carboxylating anaplerotic enzyme.

L6 ANSWER 8 OF 10 HCPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1998:184754 HCPLUS

DOCUMENT NUMBER: 128:292608

TITLE: Determination of the carbon flux in the central

AUTHOR(S): metabolism of **Corynebacterium glutamicum** by 13C-isotope analysis
CORPORATE SOURCE: Marx, Achim
Inst. Biotechnologie, Forschungszentrum Juelich
G.m.b.H., Juelich, D-52425, Germany
SOURCE: Ber. Forschungszent. Juelich (1997), Juel-3459, 1-111
pp.
DOCUMENT TYPE: CODEN: FJBEE5; ISSN: 0366-0885
Report
LANGUAGE: German
AB All C fluxes of the central metab. of C. glutamicum were quantified and the role and coordination of single metabolic pathways were studied under different metabolic situations. A method based on 13C-data was established to quantify all metabolite fluxes of the central metab. Strong sensitivities were indicated between metabolic fluxes and 13C data, thus allowing the detn. of metabolite flux. When the 13C-content of the position oxalacetate C-4 was varied by the factor 2 it could be shown if anaplerotic prodn. of C4-bodies was via the carboxylation of C3-bodies or via the glyoxalate cycle. A hyperbolic relationship was shown for the bi-directional turnover of transketolase and the 13C-content of the position pentose-5-phosphate C-1 and for the bi-directional metabolite flux between C3-bodies of glycolysis and C4-bodies of the tricarboxylate (TCA) cycle and 13C-enrichment of the position oxalacetate C-2. The NADPH balance showed that, depending on the conditions, more NADPH was produced than necessary for the synthesis of biomass and products. The NADPH excess was 16-67% in relation to the glucose uptake rate. Depending on the metabolic situation, the C4-body-decarboxylation was 10-132% and opposed to the carboxylation of C3-bodies for the anaplerotic supply of the TCA cycle. C4-body-decarboxylation and NADPH-excess as adaptations to high prodn. of Lys were minimal, with a yield coeff. of 0.32 molLysmolglucose-1. The contribution of malate enzyme to a total NADPH prodn. of 211% was small. The pentose phosphate pathway (PPP) and the TCA cycle produced 3/4 and 1/4, resp., of the total NADPH. Overexpression of glutamate dehydrogenase in a mutant of strain MH20-22B resulted in low TCA cycle flux and a high metabolite flux through the oxidative PPP. A high TCA cycle flux was detected during glutamate prodn. using strain LE4. The PPP flux was low in this strain. In a mutant of strain MH20-22B producing Lys and using NADH for synthesis of glutamate, TCA cycle flux was 79% and that of PPP was 26%. The low PPP was due to low NADPH consumption and high NADPH prodn. from isocitrate dehydrogenase of the TCA cycle. A strain ATCC 13032 isocitrate dehydrogenase mutant with a blocked TCA cycle showed a PPP flux of 62%. This mutant showed a glyoxalate cycle active in vivo when metabolizing glucose. This metabolite flux was 53%. A flux of 16% produced anaplerotically C4-bodies. At a flux of 37% the glyoxalate cycle released CO₂ by C4-body decarboxylation and pyruvate dehydrogenase.

L6 ANSWER 9 OF 10 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 94314161 MEDLINE
DOCUMENT NUMBER: 94314161 PubMed ID: 8039653
TITLE: Nucleotide sequence of the gene encoding the
Corynebacterium glutamicum mannose enzyme
II and analyses of the deduced protein sequence.
AUTHOR: Lee J K; Sung M H; Yoon K H; Yu J H; Oh T K
CORPORATE SOURCE: Genetic Engineering Research Institute, Korea Institute of
Science and Technology, Yusung, Taejon, South Korea.
SOURCE: FEMS MICROBIOLOGY LETTERS, (1994 Jun 1) 119 (1-2) 137-45.
Journal code: FML; 7705721. ISSN: 0378-1097.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-L18874

ENTRY MONTH: 199408
ENTRY DATE: Entered STN: 19940905
Last Updated on STN: 19940905
Entered Medline: 19940825

AB The complete nucleotide sequence of the gene encoding the *Corynebacterium glutamicum* mannose enzyme II (EIIMan) was determined. The gene consisted of 2052 base pairs encoding a protein of 683 amino acid residues; the molecular mass of the protein subunit was calculated to be 72570 Da. The N-terminal hydrophilic domain of EIIMan showed 39.7% homology with a C-terminal hydrophilic domain of Escherichia coli glucose-specific enzyme II (EIIIGlc). Similar homology was shown between the C-terminal sequence of EIIMan and the E. coli glucose-specific enzyme III (EIIIGlc), or the EIII-like domain of Streptococcus mutans sucrose-specific enzyme II. Sequence comparison with other EIIs showed that EIIMan contained residues His-602 and Cys-28 which were homologous to the potential phosphorylation sites of EIIIGlc, or EIII-like domains, and hydrophilic domains (IIB) of several EIIs, respectively.

L6 ANSWER 10 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 1993:482699 BIOSIS
DOCUMENT NUMBER: PREV199396116299
TITLE: Characterization of phosphoenolpyruvate carboxykinase from *Corynebacterium glutamicum*.
AUTHOR(S): Jetten, Mike S. M. (1); Sinskey, Anthony J.
CORPORATE SOURCE: (1) Dep. Biol., Room 16-238, Mass. Inst. Technol., 77 Massachusetts Ave., Cambridge, MA 02139 USA
SOURCE: FEMS (Federation of European Microbiological Societies)
Microbiology Letters, (1993) Vol. 111, No. 2-3, pp. 183-188.
ISSN: 0378-1097.
DOCUMENT TYPE: Article
LANGUAGE: English

AB Phosphoenolpyruvate (PEP) carboxykinase is present in crude extracts of *Corynebacterium glutamicum* grown on both glucose and lactate. Preparation of PEP carboxykinase free from interfering PEP carboxylase and oxaloacetate decarboxylase showed an absolute dependence on divalent manganese and IDP for activity in the oxaloacetate (OAA) formation. Other diphosphate nucleotides could not substitute for IDP. The enzyme activity displayed Michaelis-Menten kinetics for the substrates PEP, IDP, KHCO₃, OAA and ITP with a K_m of 0.7 mM, 0.4 mM, 12 mM, 1.0 mM, and 0.5 mM, respectively. At the optimum pH of 6.6, 850 nmol of OAA were formed per min per mg of protein. ATP inhibited PEP carboxykinase in the OAA forming reaction for 60% at 0.1 mM, indicating that the enzyme mainly functions in gluconeogenesis.

=> s "PTS"
L7 19911 "PTS"

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(FILE 'HOME' ENTERED AT 13:57:34 ON 01 APR 2002)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCPLUS, NTIS, LIFESCI' ENTERED AT 13:57:58 ON 01 APR 2002

L1 5706 S CORYNEBACTERIUM (A)GLUTAMICUM
L2 0 S PHOSPHOENOLPYRUVATE (W)SUGAR (W)PHOSPHOTRANSFERASE?
L3 3480 S "SUGAR PHOSPHOTRANSFERASE?"
L4 11 S PHOSPHOENOLPYRUVATE
L5 16 S L1 AND L3
L6 10 DUP REM L5 (6 DUPLICATES REMOVED)
L7 19911 S "PTS"

=> s 13 and 17
L8 1406 L3 AND L7

=> s pyruvate
L9 152465 PYRUVATE

=> s 18 and 19
L10 146 L8 AND L9

=> s 11 and 110
L11 0 L1 AND L10

=> s brevibacteriumkj
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=> s brevibacterium
L12 11661 BREVIBACTERIUM

=> s 112 and 110
L13 5 L12 AND L10

=> dup rem 113
PROCESSING COMPLETED FOR L13
L14 2 DUP REM L13 (3 DUPLICATES REMOVED)

=> d 1-2 ibib ab

L14 ANSWER 1 OF 2 LIFESCI COPYRIGHT 2002 CSA
ACCESSION NUMBER: 87:51976 LIFESCI
TITLE: Phosphoenol-pyruvate:sugar
phosphotransferase systems and sugar metabolism in
Brevibacterium flavum .
AUTHOR: Mori, M.; Shioi, I.
CORPORATE SOURCE: Cent. Res. Lab., Ajinomoto Co., Ltd., Kawasaki-ku,
Kawasaki, Kanagawa 210, Japan
SOURCE: AGRIC. BIOL. CHEM., (1987) vol. 51, no. 10, pp. 2671-2678.
DOCUMENT TYPE: Journal
FILE SEGMENT: J
LANGUAGE: English
SUMMARY LANGUAGE: English
AB **Brevibacterium** flavum mutants defective in the
phosphoenolpyruvate (PEP)-dependent glucose phosphotransferase system (PTS)
were selected with high frequency by 2-deoxyglucose-resistance. Most of them (DOG super(r)) still had the fructose-PTS
and grew not only on fructose but also on glucose like the wild-type
strain. A mutant having 1/8th the fructose-PTS activity of the
wild strain but normal glucose-PTS activity was isolated as a
xylitol-resistant mutant. It grew on glucose but not on fructose. The
glucose-PTS was active on glucose, glucosamine, 2-deoxyglucose
and mannose, and slightly on methyl-alpha-glucoside and
N-acetylglycosamine, but not on fructose or xylitol. The fructose-
PTS acted on fructose and xylitol, and to some extent on glucose
but not on glucosamine or 2-deoxyglucose. Mutants unable to grow on
glucose (DOG super(r)Glc super(-)) derived from a DOG super(r) mutant were
all defective in the fructose-PTS.

L14 ANSWER 2 OF 2 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1
ACCESSION NUMBER: 1987:277727 BIOSIS
DOCUMENT NUMBER: BA84:18766

TITLE: PYRUVATE FORMATION AND SUGAR METABOLISM IN AN AMINO ACID-PRODUCING BACTERIUM **BREVIBACTERIUM**-FLAVUM.
AUTHOR(S): MORI M; SHIIIO I
CORPORATE SOURCE: CENT. RES. LAB., AJINOMOTO CO. INC., KAWASAKI-KU, KAWASAKI, KANAGAWA 210, JPN.
SOURCE: AGRIC BIOL CHEM, (1987) 51 (1), 129-138.
CODEN: ABCHA6. ISSN: 0002-1369.

FILE SEGMENT: BA; OLD
LANGUAGE: English

AB A **Brevibacterium** flavum mutant lacking **pyruvate** kinase, No. 70, grew on glucose, fructose and sucrose as well as the original wild strain did, but was unable to grow on ribose or gluconate unless **pyruvate** was added. Mutants that required **pyruvate** for growth on ribose were derived directly from the wild strain. Many of them were completely or partially defective in **pyruvate** kinase activity. These **pyruvate** kinase mutants were also unable to grow on gluconate. A phosphoenolpyruvate (PEP): **sugar phosphotransferase** system (**PTS**) was found in *B. flavum*, which catalyzed the formation of **pyruvate** and sugar phosphate from PEP and sugar. The system required Mg²⁺, acted on glucose, fructose, mannose, glucosamine and 2-deoxyglucose, and existed in the cells grown on any of the carbon sources tested. Cells grown on fructose, mannitol and sucrose, however, exhibited higher **PTS** activities on fructose than those grown on others. Glucose **PTS** activity was about 20-fold stronger than that of glucokinase. Other sugar metabolic enzymes, inducible mannitol dehydrogenase, gluconokinase, ribokinase and maltase, as well as constitutive invertase were also detected. Oxaloacetate decarboxylase and malic enzyme, which also catalyzed the **pyruvate** formation, were found in *B. flavum*, but the latter activity was very low in cells grown on glucose. The levels of these enzymes in **pyruvate** kinase mutants unable to grow on ribose or gluconate derived from the wild strain were almost identical to those in the wild-type strain.

=> d his

(FILE 'HOME' ENTERED AT 13:57:34 ON 01 APR 2002)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCPLUS, NTIS, LIFESCI' ENTERED AT 13:57:58 ON 01 APR 2002

L1 5706 S CORYNEBACTERIUM (A) GLUTAMICUM
L2 0 S PHOSPHOENOLPYRUVATE (W) SUGAR (W) PHOSPHOTRANSFERASE?
L3 3480 S "SUGAR PHOSPHOTRANSFERASE?"
L4 11 S PHOSPHOENOLPYRUVATE
L5 16 S L1 AND L3
L6 10 DUP REM L5 (6 DUPLICATES REMOVED)
L7 19911 S "PTS"
L8 1406 S L3 AND L7
L9 152465 S PYRUVATE
L10 146 S L8 AND L9
L11 0 S L1 AND L10
L12 11661 S BREVIBACTERIUM
L13 5 S L12 AND L10
L14 2 DUP REM L13 (3 DUPLICATES REMOVED)

=> s l3 and "pep"
L15 473 L3 AND "PEP"

=> s l15 and l7
L16 280 L15 AND L7

=> s 19 and 116
L17 48 L9 AND L16

=> dup rem 117
PROCESSING COMPLETED FOR L17
L18 24 DUP REM L17 (24 DUPLICATES REMOVED)

=> d 1-24 ibib ab

L18 ANSWER 1 OF 24 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 2002:120013 SCISEARCH
THE GENUINE ARTICLE: 517LH
TITLE: Enzyme I: The gateway to the bacterial
phosphoenolpyruvate: **Sugar phosphotransferase** system
AUTHOR: Ginsburg A (Reprint); Peterkofsky A
CORPORATE SOURCE: NHLBI, Biochem Lab, Sect Prot Chem, NIH, Bldg 50, Room
2339, MSC-8012, Bethesda, MD 20892 USA (Reprint); NHLBI,
Biochem Lab, Sect Prot Chem, NIH, Bethesda, MD 20892 USA;
NHLBI, Cell Biol Lab, NIH, Bethesda, MD 20892 USA
COUNTRY OF AUTHOR: USA
SOURCE: ARCHIVES OF BIOCHEMISTRY AND BIOPHYSICS, (15 JAN 2002)
Vol. 397, No. 2, pp. 273-278.
Publisher: ACADEMIC PRESS INC, 525 B ST, STE 1900, SAN
DIEGO, CA 92101-4495 USA.
ISSN: 0003-9861.

DOCUMENT TYPE: General Review; Journal
LANGUAGE: English
REFERENCE COUNT: 38

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Regulatory aspects of the bacterial phosphoenolpyruvate (**PEP**)
:sugar **phosphotransferase** system (**PTS**) are
reviewed. The structure and conformational stability of the first protein
(enzyme I) of the **PTS**, as well as the requirement for enzyme I
to dimerize for autophosphorylation by **PEP** in the presence of
MgCl₂ are discussed. (C) 2001 Elsevier Science.

L18 ANSWER 2 OF 24 HCPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:64731 HCPLUS
DOCUMENT NUMBER: 134:248532
TITLE: Evidence of multiple regulatory functions for the PtsN
(IIANtr) protein of *Pseudomonas putida*
AUTHOR(S): Cases, Ildefonso; Lopez, Juan-Antonio; Albar,
Juan-Pablo; De Lorenzo, Victor
CORPORATE SOURCE: Centro Nacional de Biotecnologia CSIC, Madrid, 28049,
Spain
SOURCE: Journal of Bacteriology (2001), 183(3), 1032-1037
CODEN: JOBAAY; ISSN: 0021-9193
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The ptsN gene of *Pseudomonas putida* encodes IIANtr, a protein of the
phosphoenol **pyruvate:sugar phosphotransferase**
(**PTS**) system which is required for the C source inhibition of
the .sigma.54-dependent promoter Pu of the TOL (toluate degrdn.) plasmid
pWW0. Using two-dimensional gel electrophoresis, we have examd. the
effect of ptsN disruption on the general expression pattern of *P. putida*.
To this end, cells were grown in the presence or absence of glucose, and a
1,117-spot subset of the *P. putida* proteome was used as a ref. for
comparisons. Among all gene products whose expression was lowered by this
carbon source (247 spots [about 22%]), only 6 behaved as Pu (i.e., were

depressed in the ptsN background). This evidenced only a minor role for IIANtr in the extensive inhibition of gene expression in *P. putida* caused by glucose. However, the same expts. revealed a large incidence of glucose-independent effects brought about by the ptsN mutation. As many as 108 spots (ca. 9% of the cell products analyzed) were influenced, pos. or neg., by the loss of IIANtr. By matching this pattern with that of an rpoN::OMEGA.Km strain of *P. putida*, which lacks the .sigma.54 protein, we judge that most proteins whose expression was affected by ptsN were unrelated to the alternative sigma factor. These data suggest a role of IIANtr as a general regulator, independent of the presence of repressive carbon sources and not limited to .sigma.54-dependent genes.

REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 3 OF 24 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2000202048 MEDLINE
DOCUMENT NUMBER: 20202048 PubMed ID: 10736161
TITLE: Enzyme I of the phosphoenolpyruvate:**sugar phosphotransferase** system. In vitro intragenic complementation: the roles of Arg126 in phosphoryl transfer and the C-terminal domain in dimerization.
AUTHOR: Brokx S J; Talbot J; Georges F; Waygood E B
CORPORATE SOURCE: Department of Biochemistry, Health Science Building, University of Saskatchewan, Canada.
SOURCE: BIOCHEMISTRY, (2000 Apr 4) 39 (13) 3624-35.
JOURNAL code: A0G; 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200004
ENTRY DATE: Entered STN: 20000512
Last Updated on STN: 20000512
Entered Medline: 20000428

AB Enzyme I mutants of the *Salmonella typhimurium* phosphoenolpyruvate:**sugar phosphotransferase** system (PTS), which show in vitro intragenic complementation, have been identified as Arg126Cys (strain SB1690 ptsI34), Gly356Ser (strain SB1681 ptsI16), and Arg375Cys (strain SB1476 ptsI17). The mutation Arg126Cys is in the N-terminal HPr-binding domain, and complements Gly356Ser and Arg375Cys enzyme I mutations located in the C-terminal phosphoenolpyruvate(**PEP**)-binding domain. Complementation results in the formation of unstable heterodimers. None of the mutations alters the K(m) for HPr, which is phosphorylated by enzyme I. Arg126 is a conserved residue; the Arg126Cys mutation gives a V(max) of 0.04% wild-type, establishing a role in phosphoryl transfer. The Gly356Ser and Arg375Cys mutations reduce enzyme I V(max) to 4 and 2%, respectively, and for both, the **PEP** K(m) is increased from 0.1 to 3 mM. It is concluded that this activity was from the monomer, rather than the dimer normally found in assays of wild-type. In the presence of Arg126Cys enzyme, V(max) for Gly356Ser and Arg375Cys enzymes I increased 6- and 2-fold, respectively; the K(m) for **PEP** decreased to <10 &mgr;M, but the K(m) became dependent upon the stability of the heterodimer in the assay. Gly356 is conserved in enzyme I and **pyruvate** phosphate dikinase, which is a homologue of enzyme I, and this residue is part of a conserved sequence in the subunit interaction site. Gly356Ser mutation impairs enzyme I dimerization. The mutation Arg375Cys also impairs dimerization, but the equivalent residue in **pyruvate** phosphate dikinase is not associated with the subunit interaction site. A 37 000 Da, C-terminal domain of enzyme I has been expressed and purified; it dimerizes and complements Gly356Ser and Arg375Cys enzymes I proving that the association/dissociation properties of enzyme I are a function of the

C-terminal domain.

L18 ANSWER 4 OF 24 HCPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:262337 HCPLUS
DOCUMENT NUMBER: 133:116249
TITLE: ADP Modulates the Dynamic Behavior of the Glycolytic Pathway of Escherichia coli
AUTHOR(S): Diaz Ricci, Juan C.
CORPORATE SOURCE: Departamento de Bioquimica de la Nutricion, Instituto Superior de Investigaciones Biologicas (CONICET-UNT), Instituto de Quimica Biologica "Dr. Bernabe Bloj," Facultad de Bioquimica, Quimica y Farmacia, Universidad Nacional de Tucuman, Tucuman, 4000, Argent.
SOURCE: Biochemical and Biophysical Research Communications (2000), 271(1), 244-249
CODEN: BBRCA9; ISSN: 0006-291X
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A math. model that includes biochem. interactions among the **PTS** system, phosphofructokinase (PFK), and **pyruvate** kinase (PK) is used to evaluate the dynamic behavior of the glycolytic pathway of Escherichia coli under steady-state conditions. The influence of ADP, phosphoenolpyruvate (**PEP**), and fructose-6-phosphate (F6P) on the dynamic regulation of this pathway is also analyzed. The model shows that the dynamic behavior of the system is affected significantly depending on whether ADP, **PEP**, or F6P is considered const. at steady state. Sustained oscillations are obsd. only when $dADP/dt \neq 0$ and completely suppressed if $dADP/dt = 0$ at any steady-state value. However, when **PEP** or F6P is const., the system evolves toward the formation of stable limit cycles with periods ranging from 0.2 min to hours. (c) 2000 Academic Press.
REFERENCE COUNT: 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 5 OF 24 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1999042176 MEDLINE
DOCUMENT NUMBER: 99042176 PubMed ID: 9822815
TITLE: Inducer exclusion in Escherichia coli by non-**PTS** substrates: the role of the **PEP** to **pyruvate** ratio in determining the phosphorylation state of enzyme IIAGlc.
AUTHOR: Hogema B M; Arents J C; Bader R; Eijkemans K; Yoshida H; Takahashi H; Aiba H; Postma P W
CORPORATE SOURCE: E.C. Slater Institute, BioCentrum, University of Amsterdam, Plantage Muidergracht 12, 1018 TV Amsterdam, The Netherlands.
SOURCE: MOLECULAR MICROBIOLOGY, (1998 Nov) 30 (3) 487-98.
PUB. COUNTRY: Journal code: MOM; 8712028. ISSN: 0950-382X.
ENGLAND: United Kingdom
LANGUAGE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: English
ENTRY MONTH: Priority Journals
199901
ENTRY DATE: Entered STN: 19990115
Last Updated on STN: 19990115
Entered Medline: 19990104
AB The main mechanism causing catabolite repression in Escherichia coli is the dephosphorylation of enzyme IIAGlc, one of the enzymes of the phosphoenolpyruvate:carbohydrate phosphotransferase system (**PTS**). The **PTS** is involved in the uptake of a large number of

carbohydrates that are phosphorylated during transport, phosphoenolpyruvate (**PEP**) being the phosphoryl donor. Dephosphorylation of enzyme IIAGlc causes inhibition of uptake of a number of non-**PTS** carbon sources, a process called inducer exclusion. In this paper, we show that dephosphorylation of enzyme IIAGlc is not only caused by the transport of **PTS** carbohydrates, as has always been thought, and that an additional mechanism causing dephosphorylation exists. Direct monitoring of the phosphorylation state of enzyme IIAGlc also showed that many carbohydrates that are not transported by the **PTS** caused dephosphorylation during growth. In the case of glucose 6-phosphate, it was shown that transport and the first metabolic step are not involved in the dephosphorylation of enzyme IIAGlc, but that later steps in the glycolysis are essential. Evidence is provided that the [**PEP**]-[**pyruvate**] ratio, the driving force for the phosphorylation of the **PTS** proteins, determines the phosphorylation state of enzyme IIAGlc. The implications of these new findings for our view on catabolite repression and inducer exclusion are discussed.

L18 ANSWER 6 OF 24 HCPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1998:393650 HCPLUS

DOCUMENT NUMBER: 129:158337

TITLE: Identification of peptides inhibiting enzyme I of the bacterial phosphotransferase system using combinatorial cellulose-bound peptide libraries

AUTHOR(S): Mukhija, Seema; Germeroth, Lothar; Schneider-Mergener, Jens; Erni, Bernhard

CORPORATE SOURCE: Departement fur Chemie und Biochemie, Universitat Bern, Bern, Switz.

SOURCE: Eur. J. Biochem. (1998), 254(2), 433-438
CODEN: EJBCAI; ISSN: 0014-2956

PUBLISHER: Springer-Verlag

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The phosphoenolpyruvate(**P-pyruvate**)-dependent **sugar phosphotransferase** system (**PTS**) is a transport and signal-transduction system which is almost ubiquitous in bacteria but does not occur in eukaryotes. It catalyzes the uptake and phosphorylation of carbohydrates and is involved in signal transduction, e.g. catabolite repression, chemotaxis, and allosteric regulation of metabolic enzymes and transporters. EI (Enzyme I of the **PTS**) is the first and central component of the divergent **PTS** (**P-pyruvate**-dependent **sugar phosphotransferase** system) phosphorylation cascade. Using immobilized combinatorial peptide libraries and phosphorimaging, heptapeptides and octapeptides were identified which selectively inhibit EI in vitro. The IC50 of the best peptides is 30 .mu.M which is close to the KM (6 .mu.M) of EI for its natural substrate HPr (histidine contg. phosphoryl carrier protein of the **PTS**). The affinity-selected peptides are better inhibitors than a peptide with the active-site sequence of HPr. The selected peptides contain several basic residues and one arom. residue which do not occur in the active site of HPr. The large proportion of basic residues most likely reflects charge complementarity to the strongly acidic active-site pocket of EI. Guanidino groups might facilitate by complexation of the phosphoryl group the slow phosphorylation of the peptide.

L18 ANSWER 7 OF 24 MEDLINE

DUPLICATE 3

ACCESSION NUMBER: 1998143423 MEDLINE

DOCUMENT NUMBER: 98143423 PubMed ID: 9484892

TITLE: Control of the expression of the manXYZ operon in Escherichia coli: Mlc is a negative regulator of the mannose **PTS**.

AUTHOR: Plumbridge J
CORPORATE SOURCE: Institut de Biologie Physico-chimique (UPR9073), Paris,
France.. plumbridge@ibpc.fr
SOURCE: MOLECULAR MICROBIOLOGY, (1998 Jan) 27 (2) 369-80.
Journal code: MOM; 8712028. ISSN: 0950-382X.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-D90825
ENTRY MONTH: 199803
ENTRY DATE: Entered STN: 19980410
Last Updated on STN: 19980410
Entered Medline: 19980331

AB The manXYZ operon of Escherichia coli encodes a sugar transporter of the phosphoenol **pyruvate (PEP)**-dependent phosphotransferase system, which is capable of transporting many sugars, including glucose, mannose and the aminosugars, glucosamine and N-acetylglucosamine. Transcription of manX is strongly dependent on cyclic AMP (cAMP)/cAMP receptor protein (CAP). A cAMP/CAP binding site is located at -40.5, and activation by cAMP/CAP is shown to be typical of a class II promoter. The 5' end of a transcript, potentially encoding two proteins, is expressed divergently from the manXYZ operon. Previously, two binding sites for the NagC repressor were detected upstream of manX, but a mutation in nagC has very little effect on manX expression. However, a mutation in the mlc gene, encoding a homologue of nagC, results in a threefold derepression of manX expression, suggesting that this protein is a more important regulator of manX expression than NagC. The Mlc protein binds to the NagC operators, binding preferentially to the promoter-proximal operator. Plasmids overproducing either the NagC protein or the Mlc protein repress the expression of manX, but the effect of the Mlc protein is stronger. The mlc gene is shown to be allelic with the previously characterized dgsA mutation affecting the mannose phosphoenolpyruvate-dependent phosphotransferase system (**PTS**).

L18 ANSWER 8 OF 24 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 96434331 MEDLINE
DOCUMENT NUMBER: 96434331 PubMed ID: 8805571
TITLE: The first step in sugar transport: crystal structure of the amino terminal domain of enzyme I of the E. coli
PEP: sugar phosphotransferase
system and a model of the phosphotransfer complex with HPr.
AUTHOR: Liao D I; Silverton E; Seok Y J; Lee B R; Peterkofsky A;
Davies D R
CORPORATE SOURCE: Laboratory of Molecular Biology, NIDDK, National Institutes
of Health, Bethesda, MD 20892, USA.
SOURCE: STRUCTURE, (1996 Jul 15) 4 (7) 861-72.
Journal code: B31; 9418985. ISSN: 0969-2126.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-L15191; GENBANK-L46341; GENBANK-M10425;
GENBANK-M81756; GENBANK-M98359; GENBANK-U12340;
GENBANK-U15110; GENBANK-Z37113; SWISSPROT-P12654;
SWISSPROT-P23388; SWISSPROT-P23533; SWISSPROT-P23536
ENTRY MONTH: 199704
ENTRY DATE: Entered STN: 19970507
Last Updated on STN: 19970507
Entered Medline: 19970430
AB BACKGROUND: The bacterial phosphoenolpyruvate (**PEP**):
sugar phosphotransferase system (PTS)

transports exogenous hexose sugars through the membrane and tightly couples transport with phosphoryl transfer from **PEP** to the sugar via several phosphoprotein intermediates. The phosphate group is first transferred to enzyme I, second to the histidine-containing phosphocarrier protein HPr, and then to one of a number of sugar-specific enzymes II. The structures of several HPrs and enzymes IIA are known. Here we report the structure of the N-terminal half of enzyme I from Escherichia coli (EIN). RESULTS: The crystal structure of EIN (MW approximately 30 kDa) has been determined and refined at 2.5 Å resolution. It has two distinct structural subdomains; one contains four alpha helices arranged as two hairpins in a claw-like conformation. The other consists of a beta sandwich containing a three-stranded antiparallel beta sheet and a four-stranded parallel beta sheet, together with three short alpha helices. Plausible models of complexes between EIN and HPr can be made without assuming major structural changes in either protein. CONCLUSIONS: The alpha/beta subdomain of EIN is topologically similar to the phosphohistidine domain of the enzyme **pyruvate** phosphate dikinase, which is phosphorylated by **PEP** on a histidyl residue but does not interact with HPr. It is therefore likely that features of this subdomain are important in the autophosphorylation of enzyme I. The helical subdomain of EIN is not found in **pyruvate** phosphate dikinase; this subdomain is therefore more likely to be involved in phosphoryl transfer to HPr.

L18 ANSWER 9 OF 24 MEDLINE
ACCESSION NUMBER: 97141345 MEDLINE
DOCUMENT NUMBER: 97141345 PubMed ID: 8987689
TITLE: A direct comparison of approaches for increasing carbon flow to aromatic biosynthesis in Escherichia coli.
AUTHOR: Gosset G; Yong-Xiao J; Berry A
CORPORATE SOURCE: Instituto de Biotecnologia, Universidad Nacional Autonoma de Mexico, Cuernavaca, Morelos, Mexico.
SOURCE: JOURNAL OF INDUSTRIAL MICROBIOLOGY, (1996 Jul) 17 (1)
47-52.
JOURNAL code: ALF; 8610887. ISSN: 0169-4146.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: B
ENTRY MONTH: 199701
ENTRY DATE: Entered STN: 19970219
Last Updated on STN: 19970219
Entered Medline: 19970130
AB Different approaches to increasing carbon commitment to aromatic amino acid biosynthesis were compared in isogenic strains of Escherichia coli. In a strain having a wild-type **PEP**:glucose phosphotransferase (**PTS**) system, inactivation of the genes encoding **pyruvate** kinase (pykA and pykF) resulted in a 3.4 fold increase in carbon flow to aromatic biosynthesis. In a strain already having increased carbon flow to aromatics by virtue of overexpression of the tktA gene (encoding transketolase), the pykA and/or pykf mutations had no effect. A **PTS-** glucose+ mutant showed a 1.6-fold increase in carbon flow to aromatics compared to the **PTS+** control strain. In the **PTS-** glucose+ host background, overexpression of tktA caused a further 3.7-fold increase in carbon flow, while inactivation of pykA and pykF caused a 5.8-fold increase. When all of the variables tested (**PTS**-glucose+, pykA, pykF, and overexpressed tktA) were combined in a single strain, a 19.9-fold increase in carbon commitment to aromatic biosynthesis was achieved.

L18 ANSWER 10 OF 24 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 96:70638 SCISEARCH

THE GENUINE ARTICLE: TP313
TITLE: CURRENT RESEARCH ON THE GENETICS OF LACTIC-ACID PRODUCTION
IN LACTIC-ACID BACTERIA
AUTHOR: DAVIDSON B E (Reprint); LLANOS R M; CANCELLA M R; REDMAN N
C; HILLIER A J
CORPORATE SOURCE: UNIV MELBOURNE, DEPT BIOCHEM, PARKVILLE, VIC 3052,
AUSTRALIA (Reprint); CSIRO, DIV FOOD SCI & TECHNOL, DAIRY
RES LAB, HIGHETT, VIC 3190, AUSTRALIA
COUNTRY OF AUTHOR: AUSTRALIA
SOURCE: INTERNATIONAL DAIRY JOURNAL, (1995) Vol. 5, No. 8, pp.
763-784.
ISSN: 0958-6946.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: AGRI
LANGUAGE: ENGLISH
REFERENCE COUNT: 101

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Lactic acid derived from lactose is a major by-product of energy production in lactic acid bacteria. The uptake of lactose by these organisms is mediated either by the lactose phosphoenolpyruvate-phosphotransferase system (lactose **PEP-PTS**), or by a lactose-proton symport system. The disaccharide is then converted to lactate with the concomitant production of ATP. In *Lactococcus lactis* the genes encoding the lactose **PEP-PTS**, phospho-beta-galactosidase and the tagatose 6-phosphate pathway enzymes are plasmid encoded, while other genes required for lactate synthesis, including those of the Embden-Meyerhof pathway, are on the chromosome. We have compiled a current list of genes required for lactate synthesis in the lactic acid bacteria that have been cloned and characterized and discuss the present status of genetic research in this area. The analyses of the *L. lactis* lac operon have yielded one of the most detailed pictures of genetic regulation in the bacterium. The operon has been fully sequenced, the regulatory protein LacR which represses lac operon transcription has been purified and its properties determined, and the operon promoters and operators have been identified. Investigations of chromosomally encoded *L. lactis* genes have resulted in the identification and characterization of pfk, pyk, idh, tpi, and gap, which encode phosphofructokinase, **pyruvate** kinase, L-(+)-lactate dehydrogenase, triosephosphate isomerase and glyceraldehyde-3-phosphate dehydrogenase, respectively. All of these enzymes (except triosephosphate isomerase) are known from previous studies to be important in metabolite level regulation of the pathway. pfk, pyk and idh are organized into a tricistronic operon (the las operon), while tpi and gap are in monocistronic units. The las operon is so far unique to *L. lactis*. A number of investigators have studied the effect of gene dosage on glycolytic flux in lactic acid bacteria and their results are reviewed. We have introduced multiple copies of pfk, pyk, idh and the las operon into *L. lactis* and report the effect of the increase in gene dosage on enzyme levels and the rate of lactic acid production.

L18 ANSWER 11 OF 24 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 95093612 MEDLINE
DOCUMENT NUMBER: 95093612 PubMed ID: 8000534
TITLE: Vesicles prepared from *Streptococcus mutans* demonstrate the presence of a second glucose transport system.
AUTHOR: Buckley N D; Hamilton I R
CORPORATE SOURCE: Department of Oral Biology, Faculty of Dentistry,
University of Manitoba, Winnipeg, Canada.
SOURCE: MICROBIOLOGY, (1994 Oct) 140 (Pt 10) 2639-48.
Journal code: BXW; 9430468. ISSN: 1350-0872.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199501
ENTRY DATE: Entered STN: 19950215
Last Updated on STN: 19950215
Entered Medline: 19950120

AB Streptococcus mutans, an important aetiological agent of dental caries, is known to transport glucose via the phosphoenolpyruvate (**PEP**) phosphotransferase system (**PTS**). An alternative non-**PTS** glucose transport system in S. mutans Ingbratt was suggested by the increased ATP-dependent phosphorylation of glucose and the presence of higher cellular concentrations of free glucose in cells grown in continuous culture under **PTS**-repressed conditions compared to those resulting in optimal **PTS** activity. A method was developed for the preparation of membrane vesicles in order to study this system in the absence of **PTS** activity. These vesicles had very low activity of the cytoplasmic enzymes, glucokinase, **pyruvate** kinase and lactate dehydrogenase. This, coupled with the lack of glycolytic activity and the inability to transport glucose, suggested that the vesicles would also be deficient in **PTS** activity because of the absence of the general soluble **PTS** proteins, Enzyme I and HPr, required for the transport of all **PTS** sugars. Freeze-fracture electron microscopy and membrane H(+)-ATPase analysis indicated that over 90% of the vesicles had a right-side-out orientation. Vesicles from cells grown in continuous culture under **PTS**-dominant and **PTS**-repressed conditions both exhibited glucose counterflow. This indicates the presence of a constitutive non-**PTS** carrier in the organism capable of transporting glucose and utilizing ATP for glucose phosphorylation. Analysis of growth yields of cells grown under **PTS**-repressed and **PTS**-optimal conditions suggests that ATP, or an equivalent high energy molecule, must be involved in the actual transport process. This analysis is consistent with an ATP-binding protein model such as the Msm transport system reported by R. R. B. Russell and coworkers (J Biol Chem 267, 4631-4637), but it does not exclude the possibility of a separate permease for glucose.

L18 ANSWER 12 OF 24 MEDLINE
ACCESSION NUMBER: 93393384 MEDLINE
DOCUMENT NUMBER: 93393384 PubMed ID: 8216508
TITLE: Transport and metabolism of glucose and arabinose in *Bifidobacterium breve*.
AUTHOR: Degnan B A; Macfarlane G T
CORPORATE SOURCE: Medical Research Council, Dunn Clinical Nutrition Centre, Cambridge, UK.
SOURCE: ARCHIVES OF MICROBIOLOGY, (1993) 160 (2) 144-51.
Journal code: 7YN; 0410427. ISSN: 0302-8933.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199310
ENTRY DATE: Entered STN: 19931105
Last Updated on STN: 19931105
Entered Medline: 19931021

AB Glucose was required for the transport of arabinose into *Bifidobacterium breve*. The non-metabolisable glucose analogue 2-deoxy-D-glucose (2-DG) did not facilitate assimilation of arabinose. Studies using D-[U-14C]-labelled arabinose showed that it was fermented to **pyruvate**, formate, lactate and acetate, whereas the principal metabolic products of D-[U-14C]-labelled glucose were acetate and formate. In contrast to glucose, arabinose was not incorporated into cellular macromolecules. A variety of metabolic inhibitors and inhibitors of sugar transport (proton

ionophores, metal ionophores, compounds associated with electron transport) were used to investigate the mechanisms of sugar uptake. Only NaF, an inhibitor of substrate level phosphorylation, and 2-DG inhibited glucose assimilation. 2-DC had no effect on arabinose uptake, but NaF was stimulatory. High levels of phosphorylation of glucose and 2-DC by PEP and to a lesser degree, ATP were seen in phosphoenolpyruvate: phosphotransferase (PEP:PTS) assays. These data together with strong inhibition of glucose uptake by NaF suggest a role for phosphorylation in the transport process. Arabinose uptake in *B. breve* was not directly dependent on phosphorylation or any other energy-linked form of transport but may be assimilated by glucose-dependent facilitated diffusion.

L18 ANSWER 13 OF 24 MEDLINE
ACCESSION NUMBER: 92269758 MEDLINE
DOCUMENT NUMBER: 92269758 PubMed ID: 1534139
TITLE: A novel mutation FruS, altering synthesis of components of the phosphoenolpyruvate: fructose phosphotransferase system in *Escherichia coli* K12.
AUTHOR: Bolshakova T N; Molchanova M L; Erlagaeva R S; Grigorenko Y A; Gershmanovitch V N
CORPORATE SOURCE: N.F. Gamaleya Institute for Epidemiology and Microbiology, Academy of Medical Sciences, Moscow, USSR.
SOURCE: MOLECULAR AND GENERAL GENETICS, (1992 Apr) 232 (3) 394-8.
Journal code: NGP; 0125036. ISSN: 0026-8925.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199206
ENTRY DATE: Entered STN: 19920710
Last Updated on STN: 19920710
Entered Medline: 19920619

AB A novel mutation, FruS localised in the fru operon was obtained. It uncouples expression of the genes determining synthesis of the fructose-specific transport proteins and fructose-1-phosphate kinase. In FruS bacteria the fruA and fruF genes (coding for Enzyme IIfru and FPr, respectively) are constitutive by expressed while fruK (encoding fructose-1-phosphate kinase) remains inducible. In contrast to other mutations, which render expression of the whole fru operon constitutive, the FruS mutation: (1) does not lead to D-xylitol sensitivity; (2) does not inhibit growth on D-lactate, **pyruvate** and L-alanine; (3) does not decrease phosphoenolpyruvate (PEP) synthase activity.

L18 ANSWER 14 OF 24 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 93051364 MEDLINE
DOCUMENT NUMBER: 93051364 PubMed ID: 1427100
TITLE: Cloning, sequencing and expression in *Escherichia coli* of the ptsI gene encoding enzyme I of the phosphoenolpyruvate: sugar phosphotransferase transport system from *Streptococcus salivarius*.
AUTHOR: Gagnon G; Vadeboncoeur C; Levesque R C; Frenette M
CORPORATE SOURCE: Departement de Biochimie (Sciences), Universite Laval, Ste-Foy, Quebec, Canada.
SOURCE: GENE, (1992 Nov 2) 121 (1) 71-8.
Journal code: FOP; 7706761. ISSN: 0378-1119.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-M79310; GENBANK-M81114; GENBANK-M81115; GENBANK-M81116; GENBANK-M81117; GENBANK-M81118;

GENBANK-M81756; GENBANK-M95864; GENBANK-X65112;
GENBANK-X65113

ENTRY MONTH: 199212
ENTRY DATE: Entered STN: 19930122
Last Updated on STN: 19930122
Entered Medline: 19921211

AB We present the cloning and sequencing of the ptsI gene, encoding enzyme I (EI) of the phosphoenolpyruvate (**PEP**): **sugar phosphotransferase (PTS)** transport system from *Streptococcus salivarius*. The ptsI gene corresponds to an open reading frame of 1731 nucleotides, which translates into a putative 577-amino acid (aa) protein with a M(r) of 62,948 and a pI of 4.49. The EI was produced in *Escherichia coli* under the control of its own promoter located immediately upstream of ptsI, a situation never previously reported for any other gene coding for an EI. The deduced aa sequence of the *S. salivarius* EI shows a high degree of similarity with the *E. coli* EI and the EI moiety of the multiphosphoryl transfer protein from *Rhodobacter capsulatus*. The *S. salivarius* EI also shares a highly conserved aa cluster with a non-**PTS** protein, the maize **pyruvate orthophosphate dikinase**. The conserved cluster is located in a domain which is hypothesized to be the **PEP**-binding site.

L18 ANSWER 15 OF 24 HCPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1990:625291 HCPLUS
DOCUMENT NUMBER: 113:225291
TITLE: On the evolutionary origins of the bacterial
phosphoenolpyruvate:**sugar phosphotransferase** system
AUTHOR(S): Wu, L. F.; Saier, M. H., Jr.
CORPORATE SOURCE: Dep. Biol., C-016, Univ. California, San Diego, La Jolla, CA, 92093, USA
SOURCE: Mol. Microbiol. (1990), 4(7), 1219-22
CODEN: MOMIEE; ISSN: 0950-382X
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The genes encoding the proteins of the fructose-specific phosphotransferase system (**PTS**) of *Rhodobacter capsulatus* were sequenced, and the deduced amino acyl sequences of the energy-coupling protein, Enzyme I, and the transport protein, Enzyme IIfru, were compared with published sequences. Enzyme I was found to be homologous to **pyruvate:phosphate dikinase** of plants, while Enzyme IIfru was found to be homologous to the insulin-responsive glucose facilitator of mammals. The evolutionary and functional implications of these findings are discussed.

L18 ANSWER 16 OF 24 MEDLINE

ACCESSION NUMBER: 89384462 MEDLINE
DOCUMENT NUMBER: 89384462 PubMed ID: 2674659
TITLE: The repressor of the **PEP:fructose phosphotransferase** system is required for the transcription of the pps gene of *Escherichia coli*.
AUTHOR: Geerse R H; van der Pluijm J; Postma P W
CORPORATE SOURCE: E.C. Slater Institute for Biochemical Research, University of Amsterdam, The Netherlands.
SOURCE: MOLECULAR AND GENERAL GENETICS, (1989 Aug) 218 (2) 348-52.
JOURNAL code: NGP; 0125036. ISSN: 0026-8925.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198910
ENTRY DATE: Entered STN: 19900309

Last Updated on STN: 19900309
Entered Medline: 19891016

AB We have cloned the pps gene, coding for **PEP** synthase, of *Escherichia coli*. **PEP** synthase catalyses the ATP-dependent conversion of **pyruvate** into phosphoenol-**pyruvate** and is required for gluconeogenesis. The pps gene was cloned by an in vivo cloning method using a mini-Mulac bacteriophage containing a plasmid replicon. Upon expression of the cloned pps gene in the maxicell system a protein with an apparent molecular weight of 84 kDa was synthesized. The position of the pps gene of the plasmid was localized by restriction analysis of isolated transposon insertions and the determination of the **PEP** synthase activities of the different clones. An operon fusion between the pps gene and the galK gene was constructed. Measurements of the galactokinase activity in *Salmonella typhimurium* galK and galK fruR mutants showed that the transcription of the pps gene requires the presence of FruR, the repressor of the **PEP**: fructose phosphotransferase system (**PTS**) in *E. coli* and *S. typhimurium*. To test whether the components of the Fructose **PTS**, in particular FPr, are involved in the expression of the pps gene, we investigated a *S. typhimurium* galK strain, containing the fusion plasmid, in which the chromosomal fru operon was inactivated by a transposon insertion. Measurements of the galactokinase activity showed that the absence of the Fructose **PTS** proteins has no significant influence on the regulation of the pps gene.

L18 ANSWER 17 OF 24 LIFESCI COPYRIGHT 2002 CSA

ACCESSION NUMBER: 87:51976 LIFESCI
TITLE: Phosphoenol-**pyruvate:sugar**
phosphotransferase systems and sugar metabolism in *Brevibacterium flavum* .
AUTHOR: Mori, M.; Shiio, I.
CORPORATE SOURCE: Cent. Res. Lab., Ajinomoto Co., Ltd., Kawasaki-ku,
Kawasaki, Kanagawa 210, Japan
SOURCE: AGRIC. BIOL. CHEM., (1987) vol. 51, no. 10, pp. 2671-2678.
DOCUMENT TYPE: Journal
FILE SEGMENT: J
LANGUAGE: English
SUMMARY LANGUAGE: English

AB *Brevibacterium flavum* mutants defective in the phosphoenolpyruvate (**PEP**)-dependent glucose phosphotransferase system (**PTS**) were selected with high frequency by 2-deoxyglucose-resistance. Most of them (DOG super(r)) still had the fructose-**PTS** and grew not only on fructose but also on glucose like the wild-type strain. A mutant having 1/8th the fructose-**PTS** activity of the wild strain but normal glucose-**PTS** activity was isolated as a xylitol-resistant mutant. It grew on glucose but not on fructose. The glucose-**PTS** was active on glucose, glucosamine, 2-deoxyglucose and mannose, and slightly on methyl-alpha-glucoside and N-acetylglucosamine, but not on fructose or xylitol. The fructose-**PTS** acted on fructose and xylitol, and to some extent on glucose but not on glucosamine or 2-deoxyglucose. Mutants unable to grow on glucose (DOG super(r)Glc super(-)) derived from a DOG super(r) mutant were all defective in the fructose-**PTS**.

L18 ANSWER 18 OF 24 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE
7

ACCESSION NUMBER: 1987:277727 BIOSIS
DOCUMENT NUMBER: BA84:18766
TITLE: **PYRUVATE** FORMATION AND SUGAR METABOLISM IN AN AMINO ACID-PRODUCING BACTERIUM *BREVIBACTERIUM-FLAVUM*.
AUTHOR(S): MORI M; SHIIO I
CORPORATE SOURCE: CENT. RES. LAB., AJINOMOTO CO. INC., KAWASAKI-KU, KAWASAKI,
KANAGAWA 210, JPN.

SOURCE: AGRIC BIOL CHEM, (1987) 51 (1), 129-138.
CODEN: ABCHA6. ISSN: 0002-1369.

FILE SEGMENT: BA; OLD
LANGUAGE: English

AB A *Brevibacterium flavum* mutant lacking **pyruvate** kinase, No. 70, grew on glucose, fructose and sucrose as well as the original wild strain did, but was unable to grow on ribose or gluconate unless **pyruvate** was added. Mutants that required **pyruvate** for growth on ribose were derived directly from the wild strain. Many of them were completely or partially defective in **pyruvate** kinase activity. These **pyruvate** kinase mutants were also unable to grow on gluconate. A phosphoenolpyruvate (**PEP**): **sugar phosphotransferase** system (**PTS**) was found in *B. flavum*, which catalyzed the formation of **pyruvate** and sugar phosphate from **PEP** and sugar. The system required Mg²⁺, acted on glucose, fructose, mannose, glucosamine and 2-deoxyglucose, and existed in the cells grown on any of the carbon sources tested. Cells grown on fructose, mannitol and sucrose, however, exhibited higher **PTS** activities on fructose than those grown on others. Glucose **PTS** activity was about 20-fold stronger than that of glucokinase. Other sugar metabolic enzymes, inducible mannitol dehydrogenase, gluconokinase, ribokinase and maltase, as well as constitutive invertase were also detected. Oxaloacetate decarboxylase and malic enzyme, which also catalyzed the **pyruvate** formation, were found in *B. flavum*, but the latter activity was very low in cells grown on glucose. The levels of these enzymes in **pyruvate** kinase mutants unable to grow on ribose or gluconate derived from the wild strain were almost identical to those in the wild-type strain.

L18 ANSWER 19 OF 24 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1986:64690 HCAPLUS
DOCUMENT NUMBER: 104:64690
TITLE: Reconstitution of regulatory properties of adenylate cyclase in *Escherichia coli* extracts
AUTHOR(S): Reddy, Prasad; Meadow, Norman; Roseman, Saul;
Peterkofsky, Alan
CORPORATE SOURCE: Lab. Biochem. Genet., Natl. Heart, Lung Blood Inst.,
Bethesda, MD, 20892, USA
SOURCE: Proc. Natl. Acad. Sci. U. S. A. (1985), 82(24), 8300-4
CODEN: PNASA6; ISSN: 0027-8424
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The inhibition of adenylate cyclase (I) activity of *E. coli* by Me .alpha.-glucoside was demonstrated in intact or in permeable cells, but not in cell-free exts. In intact or permeable cells, this inhibition was demonstrable only in strains expressing the genes for proteins of the phosphoenolpyruvate-sugar **phosphotransferase** system (**PTS**); in permeable cells, the inhibition also requires K₂HPO₄. By using homogeneous proteins of the **PTS**, it was possible to reconstitute in cell-free exts. many of the features of the regulated form of adenylate cyclase. In the absence of K₂HPO₄, permeable cells had lower I activity than exts.; addn. of homogeneous **PTS** proteins to the exts. brought I activity close to the level obsd. in permeable cells. The low activity obsd. in permeable cells was stimulated by K₂HPO₄; this stimulation was also obsd. in exts. supplemented with **PTS** proteins and **PEP**. In permeable cells, K₂HPO₄-stimulated I activity was inhibited by Me .alpha.-glucoside or **pyruvate**; exts. behaved similarly when supplemented with **PTS** proteins, K₂HPO₄, and **PEP**. Thus, the regulated form of adenylate cyclase was reconstituted in cell-free exts. by addn. of homogeneous **PTS** proteins.

L18 ANSWER 20 OF 24 LIFESCI COPYRIGHT 2002 CSA
ACCESSION NUMBER: 85:56777 LIFESCI
TITLE: Phosphorylation of hexoses in Streptomyces aureofaciens :
Evidence that the phosphoenol-pyruvate:
sugar phosphotransferase system is not
operative.
AUTHOR: Novotna, J.; Hostalek, Z.
CORPORATE SOURCE: Inst. Microbiol., Czechoslovak Acad. Sci., 142 20 Prague 4,
Czechoslovakia
SOURCE: FEMS MICROBIOL. LETT., (1985) vol. 28, no. 3, pp. 347-350.
DOCUMENT TYPE: Journal
FILE SEGMENT: J; A
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Sugar phosphates are formed in cell-free extracts of Streptomyces aureofaciens RIA57 from glucose or fructose in the presence of phosphoenolpyruvate. In contrast to the phosphorylation by adenosine 5'-triphosphate the kinetics of formation of glucose 6-phosphate via phosphoenolpyruvate (**PEP**) is nonlinear. The product of fructose phosphorylation (only fructose 6-phosphate was determined by paper chromatography) and the absence of 1-phosphofructokinase indicate that fructose metabolism in *S. aureofaciens* does not proceed via the phosphoenolpyruvate:**sugar phosphotransferase** system (**PTS**).

L18 ANSWER 21 OF 24 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 78366270 EMBASE
DOCUMENT NUMBER: 1978366270
TITLE: The mechanism of sugar-dependent repression of synthesis of catabolic enzymes in Escherichia coli.
AUTHOR: Gonzalez J.E.; Peterkofsky A.
CORPORATE SOURCE: Lab. Biochem. Genet., Nat. Heart Lung Blood Inst., NIH, Bethesda, Md. 20014, United States
SOURCE: Progress in Clinical and Biological Research, (1978)
VOL.22/- (325-332).
CODEN: PCBRD2
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 004 Microbiology
029 Clinical Biochemistry
LANGUAGE: English
AB Previous studies have indicated that the *E. coli* adenylate cyclase (AC) activity is controlled by an interaction with the phosphoenolpyruvate (**PEP**) : **sugar phosphotransferase** system (**PTS**). A model for the regulation of AC involving the phosphorylation state of the **PTS** is described. Kinetic studies support the concept that the velocity of AC is determined by the opposing contributions of **PEP**-dependent phosphorylation (V1) and sugar-dependent dephosphorylation (V2) of the **PTS** proteins according to the expression $\% V(AC) = 100 / [1 + (\text{Max } V2/\text{Max } V1)]$. Physiological parameters influencing the rate of the **PTS** are discussed in the framework of their effects on cAMP metabolism. Factors that increase cellular concentration of **PEP** (and stimulate V1) appear to enhance AC activity while increases in extracellular sugar concentration (which stimulate V2) or internal levels of **pyruvate** (which inhibit V1) inhibit the activity of this enzyme.

L18 ANSWER 22 OF 24 MEDLINE DUPLICATE 8
ACCESSION NUMBER: 79155259 MEDLINE
DOCUMENT NUMBER: 79155259 PubMed ID: 219294
TITLE: The *Escherichia coli* adenylate cyclase complex: activation by phosphoenolpyruvate.

AUTHOR: Peterkofsky A; Gazdar C
SOURCE: JOURNAL OF SUPRAMOLECULAR STRUCTURE, (1978) 9 (2) 219-30.
Journal code: K75; 0330464. ISSN: 0091-7419.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197906
ENTRY DATE: Entered STN: 19900315
Last Updated on STN: 19980206
Entered Medline: 19790629

AB A model for the regulation of the activity of Escherichia coli adenylate cyclase is presented. It is proposed that Enzyme I of the phosphoenolpyruvate:sugar phosphotransferase system (PTS) interacts in a regulatory sense with the catalytic unit of adenylate cyclase. The phosphoenolpyruvate (PEP)-dependent phosphorylation of Enzyme I is assumed to be associated with a high activity state of adenylate cyclase. The pyruvate or sugar-dependent dephosphorylation of Enzyme I is correlated with a low activity state of adenylate cyclase. Evidence in support of the proposed model involves the observation that Enzyme I mutants have low cAMP levels and that PEP increases cellular cAMP levels and, under certain conditions, activates adenylate cyclase. Kinetic studies indicate that various ligands have opposing effects on adenylate cyclase. While PEP activates the enzyme, either glucose or pyruvate inhibit it. The unique relationships of PEP and Enzyme I to adenylate cyclase activity are discussed.

L18 ANSWER 23 OF 24 MEDLINE DUPLICATE 9
ACCESSION NUMBER: 78070166 MEDLINE
DOCUMENT NUMBER: 78070166 PubMed ID: 338995
TITLE: The mechanism of sugar-dependent repression of synthesis of catabolic enzymes in Escherichia coli.
AUTHOR: Gonzalez J E; Peterkofsky A
SOURCE: JOURNAL OF SUPRAMOLECULAR STRUCTURE, (1977) 6 (4) 495-502.
Journal code: K75; 0330464. ISSN: 0091-7419.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197802
ENTRY DATE: Entered STN: 19900314
Last Updated on STN: 19980206
Entered Medline: 19780218

AB Previous studies have indicated that the Escherichia coli adenylate cyclase (AC) activity is controlled by an interaction with the phosphoenolpyruvate (PEP): sugar phosphotransferase system (PTS). A model for the regulation of AC involving the phosphorylation state of the PTS is described. Kinetic studies support the concept that the velocity of AC is determined by the opposing contributions of PEP-dependent phosphorylation (V1) and sugar-dependent dephosphorylation (V2) of the PTS proteins according to the expression percent VAC=100/[1 + (Max V2/Max V1)]. Physiological parameters influencing the rate of the PTS are discussed in the framework of their effects on cAMP metabolism. Factors that increase cellular concentration of PEP (and stimulate V1) appear to enhance AC activity while increases in extracellular sugar concentration (which stimulate V2) or internal levels of pyruvate (which inhibit V1) inhibit the activity of this enzyme.

ACCESSION NUMBER: 77193220 EMBASE
DOCUMENT NUMBER: 1977193220
TITLE: Regulation of lactose fermentation in group N streptococci.
AUTHOR: Thomas T.D.
CORPORATE SOURCE: New Zealand Dairy Res. Inst., Palmerston North, New Zealand
SOURCE: Applied and Environmental Microbiology, (1976) 32/4
(474-478).
CODEN: AEMIDF
DOCUMENT TYPE: Journal
FILE SEGMENT: 004 Microbiology
LANGUAGE: English

AB Group N streptococci, which have the lactose phosphoenolpyruvate (**PEP**) dependent phosphotransferase system (**PTS**) and phospho .beta. D galactosidase (.beta. Pg), grew rapidly on lactose and converted more than 90% of the sugar to L lactate. In contrast, Streptococcus lactis 7962, which does not have a .beta. Pg, grew slowly on lactose and converted only 15% of the sugar to L lactate. With glucose and galactose, this strain had growth rates and fermentation patterns similar to those of other S. lactis strains, suggesting that the rapid and homolactic fermentation of lactose that is characteristic of group N streptococci is dependent upon a functional **PEP** dependent **PTS** and the presence of .beta. Pg. Seventeen strains of group N streptococci were examined for the activator specificities of **pyruvate** kinase and lactate dehydrogenase. The properties of each enzyme from all the strains, including S. lactis 7962, were similar. **Pyruvate** kinase had a broad activator specificity, whereas activation of lactate dehydrogenase was specific for ketohexose diphosphate. All intermediates of lactose metabolism from the hexose phosphates to the triose phosphates activated **pyruvate** kinase. No activation was obtained with adenosine 5' monophosphate. K⁺ and Mg²⁺ were required for **pyruvate** kinase activity but could be replaced by NH₄⁺ and Mn²⁺, respectively. Lactate dehydrogenase was activated equally by fructose 1,6 diphosphate and tagatose 1,6 diphosphate, the activation characteristics being pH dependent. The roles of **pyruvate** kinase and lactate dehydrogenase in the regulation of lactose fermentation by group N streptococci are discussed.

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FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCPLUS, NTIS,
LIFESCI' ENTERED AT 13:57:58 ON 01 APR 2002

L1 5706 S CORYNEBACTERIUM (A)GLUTAMICUM
L2 0 S PHOSPHOENOLPYRUVATE (W)SUGAR (W)PHOSPHOTRANSFERASE?
L3 3480 S "SUGAR PHOSPHOTRANSFERASE?"
L4 11 S PHOSPHOENOLPYRUVATE
L5 16 S L1 AND L3
L6 10 DUP REM L5 (6 DUPLICATES REMOVED)
L7 19911 S "PTS"
L8 1406 S L3 AND L7
L9 152465 S PYRUVATE
L10 146 S L8 AND L9
L11 0 S L1 AND L10
L12 11661 S BREVIBACTERIUM
L13 5 S L12 AND L10
L14 2 DUP REM L13 (3 DUPLICATES REMOVED)
L15 473 S L3 AND "PEP"
L16 280 S L15 AND L7
L17 48 S L9 AND L16
L18 24 DUP REM L17 (24 DUPLICATES REMOVED)

=> s l18 and l1
L19 0 L18 AND L1

=> e pompejus m/au

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| E1 | 3 | POMPEIUS P/AU |
| E2 | 42 | POMPEIUS R/AU |
| E3 | 26 | --> POMPEJUS M/AU |
| E4 | 22 | POMPEJUS MARKUS/AU |
| E5 | 37 | POMPEKIRN V/AU |
| E6 | 1 | POMPEL M/AU |
| E7 | 1 | POMPEL P/AU |
| E8 | 1 | POMPELEAN A/AU |
| E9 | 1 | POMPELIA D MICK/AU |
| E10 | 2 | POMPELIA DONALD M/AU |
| E11 | 2 | POMPELIANO O/AU |
| E12 | 214 | POMPELLA A/AU |

=> s e3
L20 26 "POMPEJUS M"/AU

=> s e3-e4
L21 48 ("POMPEJUS M"/AU OR "POMPEJUS MARKUS"/AU)

=> e kroger b/au

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| E1 | 2 | KROGER ANDREW/AU |
| E2 | 1 | KROGER ANNELENE/AU |
| E3 | 92 | --> KROGER B/AU |
| E4 | 6 | KROGER B J/AU |
| E5 | 1 | KROGER BERND/AU |
| E6 | 1 | KROGER BERNHARD/AU |
| E7 | 4 | KROGER BLOCK A/AU |
| E8 | 15 | KROGER BURKHARD/AU |
| E9 | 75 | KROGER C/AU |
| E10 | 21 | KROGER C F/AU |
| E11 | 1 | KROGER C R/AU |
| E12 | 1 | KROGER C S/AU |

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L22 92 "KROGER B"/AU

=> e schroder h/au

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| E1 | 1 | SCHRODER GUSTAV ADOLF/AU |
| E2 | 1 | SCHRODER GUY/AU |
| E3 | 1910 | --> SCHRODER H/AU |
| E4 | 45 | SCHRODER H A/AU |
| E5 | 727 | SCHRODER H C/AU |
| E6 | 5 | SCHRODER H CH/AU |
| E7 | 1 | SCHRODER H CORRECTED TO SCHODER H/AU |
| E8 | 365 | SCHRODER H D/AU |
| E9 | 1 | SCHRODER H D A A/AU |
| E10 | 1 | SCHRODER H DAA/AU |
| E11 | 98 | SCHRODER H E/AU |
| E12 | 60 | SCHRODER H F/AU |

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L23 1910 "SCHRODER H"/AU

=> e zelder o/au

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| E1 | 3 | ZELDER J/AU |
| E2 | 2 | ZELDER JOY B/AU |
| E3 | 180 | --> ZELDER O/AU |

E4 1 ZELDER OOSKAR/AU
E5 49 ZELDER OSKAR/AU
E6 1 ZELDER S/AU
E7 1 ZELDER SUSANNE/AU
E8 1 ZELDERS F/AU
E9 1 ZELDERS H G/AU
E10 2 ZELDERS I T/AU
E11 16 ZELDERS T/AU
E12 1 ZELDERS THEO/AU

=> s e3
L24 180 "ZELDER O"/AU

=> e haberhauer g/au
E1 2 HABERHAUER D/AU
E2 1 HABERHAUER F/AU
E3 197 --> HABERHAUER G/AU
E4 12 HABERHAUER GEBHARD/AU
E5 20 HABERHAUER GEORG/AU
E6 7 HABERHAUER GREGOR/AU
E7 2 HABERHAUER GUENTHER/AU
E8 1 HABERHAUER HELMUT/AU
E9 5 HABERHAUER HELMUTH/AU
E10 1 HABERHAUER OSWALD/AU
E11 12 HABERHAUER P/AU
E12 2 HABERHAUER PETER/AU

=> s e3-e6
L25 236 ("HABERHAUER G"/AU OR "HABERHAUER GEBHARD"/AU OR "HABERHAUER GEORG"/AU OR "HABERHAUER GREGOR"/AU)

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(FILE 'HOME' ENTERED AT 13:57:34 ON 01 APR 2002)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCPLUS, NTIS,
LIFESCI' ENTERED AT 13:57:58 ON 01 APR 2002

L1 5706 S CORYNEBACTERIUM (A)GLUTAMICUM
L2 0 S PHOSPHOENOLPYRUVATE (W) SUGAR (W) PHOSPHOTRANSFERASE?
L3 3480 S "SUGAR PHOSPHOTRANSFERASE?"
L4 11 S PHOSPHOENOLPYRUVATE
L5 16 S L1 AND L3
L6 10 DUP REM L5 (6 DUPLICATES REMOVED)
L7 19911 S "PTS"
L8 1406 S L3 AND L7
L9 152465 S PYRUVATE
L10 146 S L8 AND L9
L11 0 S L1 AND L10
L12 11661 S BREVIBACTERIUM
L13 5 S L12 AND L10
L14 2 DUP REM L13 (3 DUPLICATES REMOVED)
L15 473 S L3 AND "PEP"
L16 280 S L15 AND L7
L17 48 S L9 AND L16
L18 24 DUP REM L17 (24 DUPLICATES REMOVED)
L19 0 S L18 AND L1
E POMPEJUS M/AU
L20 26 S E3
L21 48 S E3-E4
E KROGER B/AU
L22 92 S E3
E SCHRODER H/AU

L23 1910 S E3
E ZELDER O/AU
L24 180 S E3
E HABERHAUER G/AU
L25 236 S E3-E6

=> s 121 -125

MISSING OPERATOR L21 -L25

The search profile that was entered contains terms or nested terms that are not separated by a logical operator.

=> s 121 or 122 or 123 or 124 or 125

L26 2444 L21 OR L22 OR L23 OR L24 OR L25

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(FILE 'HOME' ENTERED AT 13:57:34 ON 01 APR 2002)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS, LIFESCI' ENTERED AT 13:57:58 ON 01 APR 2002

L1 5706 S CORYNEBACTERIUM (A) GLUTAMICUM
L2 0 S PHOSPHOENYL PYRUVATE (W) SUGAR (W) PHOSPHOTRANSFERASE?
L3 3480 S "SUGAR PHOSPHOTRANSFERASE?"
L4 11 S PHOSPHOENYL PYRUVATE
L5 16 S L1 AND L3
L6 10 DUP REM L5 (6 DUPLICATES REMOVED)
L7 19911 S "PTS"
L8 1406 S L3 AND L7
L9 152465 S PYRUVATE
L10 146 S L8 AND L9
L11 0 S L1 AND L10
L12 11661 S BREVIBACTERIUM
L13 5 S L12 AND L10
L14 2 DUP REM L13 (3 DUPLICATES REMOVED)
L15 473 S L3 AND "PEP"
L16 280 S L15 AND L7
L17 48 S L9 AND L16
L18 24 DUP REM L17 (24 DUPLICATES REMOVED)
L19 0 S L18 AND L1
E POMPEJUS M/AU
L20 26 S E3
L21 48 S E3-E4
E KROGER B/AU
L22 92 S E3
E SCHRODER H/AU
L23 1910 S E3
E ZELDER O/AU
L24 180 S E3
E HABERHAUER G/AU
L25 236 S E3-E6
L26 2444 S L21 OR L22 OR L23 OR L24 OR L25

=> s 126 and 117

L27 0 L26 AND L17

=> s 126 and 13

L28 2 L26 AND L3

=> d 1-2 ibib ab

L28 ANSWER 1 OF 2 BIOTECHDS COPYRIGHT 2002 DERWENT INFO AND ISI
ACCESSION NUMBER: 2001-04894 BIOTECHDS

TITLE: Corynebacterium glutamicum nucleic acids encoding
 phosphoenolpyruvate:sugar
phosphotransferase system proteins or their portions,
 useful for typing or identifying C. glutamicum or related
 bacteria, and as markers for transformation;
 selectable marker

AUTHOR: Pompejus M; Kroeger B; Schroeder H; Zelder
 O; Haberhauer G

PATENT ASSIGNEE: BASF

LOCATION: Ludwigshafen, Germany.

PATENT INFO: WO 2001002583 11 Jan 2001

APPLICATION INFO: WO 2000-DE973 27 Jun 2000

PRIORITY INFO: DE 1999-1042097 3 Sep 1999; US 1999-142691 1 Jul 1999

DOCUMENT TYPE: Patent

LANGUAGE: English

OTHER SOURCE: WPI: 2001-080989 [09]

AB Isolated Corynebacterium glutamicum ATCC 13032 nucleic acids encoding
 phosphoenolpyruvate:sugar-phosphotransferase system
 (PTS) proteins or their fragments are claimed. A PTS nucleic acid (N1)
 does not consist of any of the F-designated genes defined and is selected
 from one of 17 disclosed nucleic acid sequences (S1) and their fragments
 nucleic acid which encode a protein selected from one of the 17 protein
 sequences (S2) disclosed; nucleic acid encoding a naturally occurring
 allelic variant of a protein selected from (S2). Also claimed are
 methods for producing the proteins; C. glutamicum PTS protein and its
 fragments; diagnosis of Corynebacterium diphtheriae infection; fusion
 proteins; antisense PTS nucleic acid; a method for screening molecules
 which modulate the activity of a PTS protein; and a transformed host
 cell. (144pp)

L28 ANSWER 2 OF 2 HCPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2001:31659 HCPLUS
 DOCUMENT NUMBER: 134:96287
 TITLE: Corynebacterium glutamicum genes encoding
 phosphoenolpyruvate:sugar
phosphotransferase system proteins
 INVENTOR(S): Pompejus, Markus; Kroger, Burkhard;
 Schroder, Hartwig; Zelder, Oskar; Haberhauer,
 Gregor
 PATENT ASSIGNEE(S): BASF Aktiengesellschaft, Germany
 SOURCE: PCT Int. Appl., 143 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 6
 PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|------------------------|---|-----------------|-----------------|----------|
| WO 2001002583 | A2 | 20010111 | WO 2000-IB973 | 20000627 |
| WO 2001002583 | A3 | 20010726 | | |
| W: | AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,
HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,
LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU,
ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM | | | |
| RW: | GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ,
CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG | | | |
| PRIORITY APPLN. INFO.: | | US 1999-142691P | P | 19990701 |
| | | US 1999-150310P | P | 19990823 |

DE 1999-19942095 A 19990903
DE 1999-19942097 A 19990903

AB Isolated nucleic acid mols., designated phosphoenolpyruvate:**sugar phosphotransferase** (PTS) nucleic acid mols., which encode novel PTS proteins from *Corynebacterium glutamicum* are described. The invention also provides antisense nucleic acid mols., recombinant expression vectors contg. PTS nucleic acid mols., and host cells into which the expression vectors have been introduced. The invention still further provides isolated PTS proteins, mutated PTS proteins, fusion proteins, antigenic peptides and methods for the improvement of prodn. of a desired compd. from *C. glutamicum* based on genetic engineering of PTS genes in this organism.

=> d his

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L1 5706 S CORYNEBACTERIUM (A) GLUTAMICUM
L2 0 S PHOSPHOENOLPYRUVATE (W) SUGAR (W) PHOSPHOTRANSFERASE?
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L10 146 S L8 AND L9
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L13 5 S L12 AND L10
L14 2 DUP REM L13 (3 DUPLICATES REMOVED)
L15 473 S L3 AND "PEP"
L16 280 S L15 AND L7
L17 48 S L9 AND L16
L18 24 DUP REM L17 (24 DUPLICATES REMOVED)
L19 0 S L18 AND L1
E POMPEJUS M/AU
L20 26 S E3
L21 48 S E3-E4
E KROGER B/AU
L22 92 S E3

| | Document ID | Issue Date | Pages | Title |
|---|--------------------|-------------------|--------------|--|
| 1 | US 20020032323 A1 | 20020314 | 64 | STREPTOCOCCUS PNEUMONIAE POLYNUCLEOTIDES AND SEQUENCES |
| 2 | US 6245502 B1 | 20010612 | 23 | Target system |
| 3 | US 6162627 A | 20001219 | 117 | Methods of identifying inhibitors of sensor histidine kinases through rational drug design |
| 4 | US 6077682 A | 20000620 | 126 | Methods of identifying inhibitors of sensor histidine kinases through rational drug design |

| | Document ID | Issue Date | Pages | Title |
|---|--------------|------------|-------|--|
| 1 | US 6162627 A | 20001219 | 117 | Methods of identifying inhibitors of sensor histidine kinases through rational drug design |
| 2 | US 6077682 A | 20000620 | 126 | Methods of identifying inhibitors of sensor histidine kinases through rational drug design |

| | U | 1 | Document ID | Issue Date | Pages |
|---|--------------------------|--------------------------|----------------------|------------|-------|
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A1 | 20020314 | 64 |

| | Title | Current OR | Current XRef |
|---|---|------------|--|
| 1 | STREPTOCOCCUS PNEUMONIAE
POLYNUCLEOTIDES AND SEQUENCES | 536/23.7 | 435/252.3;
435/320.1;
435/69.1;
536/24.32 |

| | Retrieval
Classif | Inventor | S | C | P | 2 | 3 | 4 | 5 |
|---|----------------------|------------------------------|-------------------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|
| 1 | | KUNSCH, CHARLES A. et
al. | <input checked="" type="checkbox"/> | <input type="checkbox"/> |

| | Image Doc.
Displayed | PT |
|---|---------------------------------|--------------------------|
| 1 | US 20020032323 | <input type="checkbox"/> |

| | L # | Hits | Search Text |
|----|-----|--------|---|
| 1 | L1 | 355 | corynebacterium adj
glutamicum |
| 2 | L2 | 0 | sugar adj
phosphotransferase\$2 |
| 3 | L4 | 0 | 11 same 13 |
| 4 | L3 | 4 | sugar adj
phosphotransferase\$2 |
| 5 | L5 | 8322 | pyruvate |
| 6 | L6 | 2 | 13 same 15 |
| 7 | L7 | 413156 | clon\$3 or express\$3 or
recombinant |
| 8 | L8 | 1 | 13 same 17 |
| 9 | L9 | 0 | pompejus.in. |
| 10 | L10 | 115 | kroger.in. |
| 11 | L11 | 703 | schroder.in. |
| 12 | L12 | 19 | haberhauer.in. |

| | L # | Hits | Search Text |
|----|-----|------|-------------------|
| 13 | L13 | 836 | 110 or 111 or 112 |
| 14 | L14 | 0 | 113 and 13 |
| 15 | L15 | 0 | 113 and 11 |